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CAPABILITY OF VIRTUAL ENVIRONMENTS TO MEET MILITARY REQUIREMENTS

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The DoD and NASA are considering virtual environment (VE) technology for use in forward deployable and remote training devices. Yet, many of these VE devices, particularly those which employ helmet-mounted displays, have an adverse effect on users, eliciting motion sickness and other sequelae (e.g., Pausch, Crea, & Conway, 1992; Kennedy, Lane, Lilienthal, Berbaum, & Hettinger, 1992). These symptoms, now called cybersickness (McCauley & Sharkey, 1992), could retard development of VE technology and limit its use as a training tool.

Motion sickness is known to be polysymptomatic and in scoring self-reports we have found there to be reliably different profiles of sickness in simulators, at sea, in space, and in VE (Kennedy, Lane, Berbaum, & Lilienthal, 1993). Furthermore, recent research in our laboratories implies that cybersickness may involve multiple functional pathways. The first pathway is related to ill-effects upon the autonomic nervous system or ANS (Money, Lackner, & Cheung, 1996). According to sensory conflict theory (Reason & Brand, 1975), the ANS is provoked when sensory inputs from the visual, auditory, vestibular, or somatoceptors are uncorrelated or incompatible. This is the case when one is exposed to the certain sensory rearrangements in a virtual environment. Such rearrangements can trigger the "emetic brain response" (Oman, 1991), causing vomiting, perspiration, nausea, pallor, salivation, and drowsiness.

Slower effects might include the sopite syndrome. This syndrome may involve a sleep-related pathway. Student Naval aviators, referred for airsickness, show that sopite syndrome can occur during military flight training in an individual who is "immune" to airsickness and debilitate an individual long after he has recovered from overt airsickness.

When the ANS pathway is triggered it can also lead to performance decrements. Performance problems can arise due to lowered arousal and decreased concentration or because individuals experiencing ANS symptoms will attempt to minimize ill-effects by modifying their behavior (Hettinger, Kennedy, & McCauley, 1990).

It has also been shown that individuals restrict their head movements while using helmet-mounted displays (Hennesy, Sharkey, Matsumoto, & Voorhees, 1992), and that inhibition of head movement "learned" in the trainer transfers to performance in the actual helicopter. It is clear that if VE systems are to be effective training devices, such negative transfer must be avoided by obtaining a better understanding of the ANS mechanism.

Another cybersickness pathway involves adaptation within the central nervous system (CNS). In a well-respected theory of CNS functioning, von Holst (1954) argues that motor impulses (or efference) leave images in the CNS which are compared to the reafference generated by the effector (i.e., the stimuli resulting from one's own muscular activity). Normally, the efference and reafference images match, thus leading to coordinated muscular activity. When something causes a mismatch, coordinated activities may be degraded (e.g., degraded hand-eye coordination) because of the limiting effects of the reafference. This mismatch can also provoke the ANS pathway, producing motion sickness. Held (1965) and Reason (1978) provide evidence to suggest that the effects of the ANS pathway discussed above can be overcome by facilitating CNS adaptation to these neural mismatches. Regardless of one's opinions about a neural mismatch hypothesis, it is clear that adaptation is fostered when "mismatches" are sufficiently regular, users have control over their movements, and/or they receive a sensory response (e.g., visual, vestibular, or proprioceptive) to their actions.

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Thus, if the sensory rearrangements of the VE are sufficiently small, gradual, regular, and "reafferent", the plasticity of the CNS more frequently allows adaption. This adaptation is characterized by: a decline in the initial response to an altered stimulus; development of an altered, often compensatory response following prolonged exposure to the change; and a continuation of the adapted response (i.e., an aftereffect) once the stimulus is removed (Dolezal, 1982; Parker & Parker, 1990; Welch, 1978). Such post-adaptation aftereffects have been known to persist for several hours after system exposure (Baltzley, Kennedy, Berbaum, Lilienthal, & Gower, 1989; Crosby & Kennedy, 1982; Unga, 1987) and may occur without any symptoms of motion sickness discomfort except disorientation.

Along the first ANS pathway of cybersickness, if an individual is sick he/she will perceive this discomfort and modify his/her behavior to minimize the ill-effects. Along the second pathway, however, if adaptive changes occur individuals may be unaware of the CNS modifications. Since the two pathways follow a different time course, it is possible for both effects to be present; or either may be present without the other. We will report some preliminary evidence for these predictions.

Much of the research to date on self-reports of sickness symptomatology has used a measure called the Motion Sickness Questionnaire (MSQ). The MSQ was developed more than thirty years ago for studying the causal mechanisms underlying motion sickness. A paper-and-pencil version of the MSQ was later tailored to collect data from simulator participants, and scoring norms were developed using a calibration sample of 1600 simulator exposures. The Simulator Sickness Questionnaire (SSQ) was developed by Kennedy, Lane, Berbaum, and Lilienthal (1993) based on empirical studies. The symptom checklist portion of the SSQ is comprised of 28 symptoms, most of which are rated on a four-point ordinal scale with anchor points at "none," "mild," "moderate," and "severe". The items on the checklist have considerable overlap with the signs and symptom checklist of Lackner and Graybiel. Scoring our checklist by their scoring key versus ours produces Total Scores from the two methods which correlate ($r > .83$) and we showed a figure of our database of two dozen simulators that we use as norms and which are scored also with the Lackner scoring key. We also presented additional normative data for 8,000 exposures from simulators to be compared with data from >450 virtual reality exposures.

In addition to the algorithm for Total Score, the original data base of 1000+ exposures was also used to carry out a factor analysis of simulator sickness symptoms (Kennedy et al., 1993). The symptoms from this large group of exposures revealed three clearly defined factors: nausea and neurovegetative complaints (N), oculomotor disturbances (O) and disorientation effects (D) (cf. also Lane & Kennedy, 1988). These three clusters fit nicely with theoretical descriptions of motion sickness (e.g., Money, 1970; Reason & Brand, 1975) and, for those of us who have personally experienced motion sickness, these three factors have obvious face validity. Compared to the Lackner and Graybiel scoring method, the nausea subscale of our scoring system correlates higher ($r = .88$) with their total score. The total score correlates much lower ($r = .62$) with our disorientation and oculomotor scoring keys. From this factor analytic approach we have therefore hypothesized that if sufficient individuals are studied, it may be possible that the distribution or configuration of the three factors may turn out to be consistent within a given simulator and different between simulators. If so, then perhaps this might provide a method whereby the many different causes of simulator sickness can be delineated. Therefore, while Total Score differences in simulators may index the level of the problem, differences in profile or configuration, REGARDLESS OF LEVEL OF SICKNESS, may signal the nature of the cause of sickness in that simulator.

In a series of 3 experiments, using 3 different scenarios displayed over two different helmet mounted systems, subjects ($N = 75$) were exposed to 20-40 minutes of VE. We found in all three studies:

- 1) pre post differences in self-report of motion sickness were statistically significant;
- 2) pre post motor effects (past pointing and/or posture changes) were significantly different;
- 3) although changes in both types of variables were reliably observed, the sickness severity and size of side effects themselves were not correlated. That means that persons who were sick may or may not have had postural effects and the converse.

These data were compared and discussed with several other cases in which different aspects of motion sickness, while perhaps not totally independent, are clearly not isomorphic. This independence of function persists generally across other situations in which motion sickness is elicited (e.g., after space flight). If so, any independent and uncorrelated

aftereffects should be assessed individually as each may have different safety implications. Furthermore, independence of function would imply different pathways and perhaps different centers of control.

In conclusion, we propose that there at least two major pathways involved in cybersickness, each exhibiting different performance and safety problems during a motion challenge. We believe that the time course of each pathway needs to be systematically measured. Such empirical knowledge may enable us to avoid triggering the ANS pathway by using brief bursts of exposure, while simultaneously promoting CNS adaptation during repeated and carefully limited exposures.

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